PERIODIC HEALTH EXAMINATION, 1995 UPDATE: 3. SCREENING FOR VISUAL PROBLEMS AMONG ELDERLY PATIENTS

Canadian Task Force on the Periodic Health Examination

Abstract • Résumé

Objective: To provide recommendations to family physicians for screening elderly patients (over 65 years of age) for visual impairment and its common clinical causes.

Options: Visual acuity screening with Snellen sight chart, funduscopy, retinal photography, tonometry and perimetry.

Outcomes: Delay or prevention of visual deterioration or blindness.

Evidence: A MEDLINE search for relevant articles published between January 1986 and December 1993 was undertaken, the bibliographies of the articles were scrutinized for additional articles, and experts were consulted. The highest available level of evidence was used in making recommendations.

Values: The evidence-based methods and values of the Canadian Task Force on the Periodic Health Examination were used. Preservation of vision was given the highest value in accordance with other guidelines regarding eyesight.

Benefits, harms and costs: Potential benefits are to maintain or improve visual acuity. Potential for harm to patients is minimal. Limited data are available on costs.

Recommendations: There is fair evidence to include in the periodic health examination visual acuity testing with a Snellen sight chart and funduscopy or retinal photography in elderly patients with diabetes of at least 5 years' duration (grade B recommendation). The place of funduscopy in the detection of age-related macular degeneration and glaucomatous changes is controversial. For patients at high risk for glaucoma (positive family history, black race, severe myopia or diabetes) it would be prudent to have a periodic assessment by an ophthalmologist.

Validation: Recommendations differ from those of the American Academy of Ophthalmology and the American Optometric Association. Recommendations for glaucoma screening are similar to those of the US Preventive Services Task Force. Present recommendations have been reviewed by experts in ophthalmology and optometry.

Sponsor: These guidelines were developed and endorsed by the task force, which is funded by Health Canada and the National Health Research and Development Program. The principal author (C.P.) was supported in part by the Educational Centre for Aging and Health, McMaster University, Hamilton, Ont.

Objectif : Fournir aux médecins de famille des recommandations sur le dépistage de l'incapacité visuelle et de ses causes cliniques communes chez les patients âgés (65 ans et plus).

__Chairman__ (up to Nov. 1, 1994): Dr. Richard Goldbloom, professor, Department of Pediatrics, Dalhousie University, Halifax, NS. __Vice-chairman__ (resigned as of Nov. 1, 1994): Dr. Renaldo N. Battista, director, Division of Clinical Epidemiology, Montreal General Hospital, Montreal, Que. __Members:__ Drs. Geoffrey Anderson, assistant professor, Department of Health Administration, University of Toronto, Toronto, Ont.; Marie-Dominique Beaulieu, associate professor, Department of Family Medicine, University of Montreal, Montreal, Que.; R. Wayne Elford, professor and chairman of research, Department of Family Medicine, University of Calgary, Calgary, Alta.; John W. Feightner (chairman of the task force as of Nov. 1, 1994), professor and research director, Department of Family Medicine, McMaster University, Hamilton, Ont.; William Feldman, professor of pediatrics, University of Toronto, and head, Division of General Pediatrics, Hospital for Sick Children, Toronto, Ont.; Alexander G. Logan (resigned as of Nov. 1, 1994), professor, Faculty of Medicine, University of Toronto, Toronto, Ont.; Brenda Morrison, professor, Department of Health Care and Epidemiology, University of British Columbia, Vancouver, BC; David Offord (resigned as of Nov. 1, 1994), professor, Department of Psychiatry, McMaster University, Hamilton, Ont.; Christopher Patterson (__principal author__), professor and head, Division of Geriatric Medicine, McMaster University, Hamilton, Ont.; Walter O. Spitzer, professor, Department of Epidemiology and Biostatistics, McGill University, Montreal, Que.; and Elaine Wang, associate professor, Department of Pediatrics, Faculty of Medicine, University of Toronto, Toronto, Ont. __Resource people:__ Dr. Philip Mickelson, medical consultant, health standards, Health Services Directorate, Health Canada, Ottawa, Ont.; and Ms. Jennifer Dingle, coordinator, Canadian Task Force on the Periodic Health Examination, Department of Pediatrics, Dalhousie University, Halifax, NS. __New members as of Nov. 1, 1994:__ Drs. Harriet MacMillan, assistant professor, departments of Pediatrics and Psychiatry, McMaster University, Hamilton, Ont.; Robin McLeod, associate professor, Department of Surgery, University of Toronto, Toronto, Ont.; and Jean-Marie Moutquin, professor, Department of Obstetrics and Gynecology, Université Laval, Quebec City, Que.

Copies of this and other task force reports are available from the Health Services Directorate, Health Programs and Services Branch, Health Canada, Tunney's Pasture, Ottawa ON K1A 1B4.

Options : Test d'acuité visuelle à l'aide de l'échelle de Snellen, examen du fond de l'oeil, photographie de la rétine, tonométrie et périmétrie.

Résultats : Retard ou prévention de la détérioration de la vue ou de la cécité.

Preuves : On a cherché, dans MEDLINE, des articles sur la question publiés entre janvier 1986 et décembre 1993, examiné les bibliographies des articles pour y trouver d'autres articles, et consulté des experts. On a fondé les recommandations sur le niveau de preuve le plus élevé disponible.

Valeurs : On a utilisé les méthodes fondées sur la preuve et les valeurs du Groupe d'étude canadien sur l'examen médical périodique. On a accordé la valeur la plus importante à la préservation de la vision conformément à d'autres lignes directrices sur la vue.

Avantages, préjudices et coûts : Les avantages possibles consistent à maintenir ou améliorer l'acuité visuelle. Les risques de préjudices pour les patients sont minimes. Les données disponibles sur les coûts sont limitées.

Recommandations : Des preuves raisonnables incitent à inclure dans l'examen médical périodique des tests d'acuité visuelle effectués à l'aide de l'échelle de Snellen et d'un examen du fond de l'oeil ou d'une photographie de la rétine chez les patients âgés atteints de diabète depuis au moins 5 ans (recommandation de catégorie B). Le rôle de l'examen du fond de l'oeil dans la détection d'une dégénérescence de la macule et de changements glaucomateux liés à l'âge suscite la controverse. Il serait prudent, pour les patients à risque élevé de glaucome (antécédents familiaux positifs, race noire, myopie grave ou de diabète), de subir un examen périodique effectué par un ophtalmologue.

Validation : Les recommandations diffèrent de celles de l'American Academy of Ophthalmology et de l'American Optometric Association. Les recommandations relatives au dépistage du glaucome sont semblables à celles du Preventive Services Task Force des États-Unis. Les recommandations en vigueur ont été examinées par des experts des domaines de l'ophtalmologie et de l'optométrie.

Commanditaire : Ces lignes directrices ont été mises au point et appuyées par le Groupe de travail, qui est financé par Santé Canada et par le Programme national de recherche et développement en matière de santé. L'auteur principal (C.P.) a reçu une partie de son appui du Centre d'éducation sur le vieillissement et la santé de l'Université McMaster, de Hamilton (Ont.).

This article is intended to provide family physicians with recommendations on screening elderly patients (over 65 years of age) for visual impairment and its common clinical causes. The evidence-based methods and values of the Canadian Task Force on the Periodic Health Examination were used.[1]

MEDLINE was searched for articles published between January 1986 and December 1993 with the use of the following headings and key words: "glaucoma (MH)," "glaucoma, suspect (MeSH) screening or vision screening (MH)," "clinical trial (PT)," "glaucoma–drug therapy," "intraocular pressure–drug effect," "timolol–administration and dosage," "vision disorders," "aged," "diabetic retinopathy," "age-related macular degeneration," "cataract" and "retinal diseases." In addition, the bibliographies of relevant articles were scrutinized for additional articles, the recommendations of other groups were reviewed, and experts in ophthalmology and optometry were consulted to obtain the highest available level of evidence to make recommendations. The highest value selected was preservation of vision, in accordance with other guidelines regarding eyesight.

Visual impairment is defined as an acuity of less than 20/60 (6/18) in the better eye, with the best correction.[2] Legal blindness is defined as an acuity of less than 20/160 (6/48) in the United States and less than 20/200 (6/60) in Canada.[2] The World Health Organization (WHO) has standardized the definitions as follows: visual impairment, acuity of less than 20/60 (6/18), and legal blindness, acuity of less than 20/400 (6/120), with the best correction in both cases.[3] People with severe visual disability are eligible to be registered legally blind, which allows for dispensations related to income tax and other benefits. Registration in Canada requires examination by an ophthalmologist.

Burden of suffering

Visual impairment of some form affects 13% of elderly people; almost 8% of them have severe impairment (blindness in both eyes or inability to read newsprint even with glasses).[4] About 1% of people over 40 years have bilateral blindness.[5] In 1989, there were 63 576 people registered as legally blind in Canada.[6] The leading causes of visual impairment in elderly people are presbyopia, cataracts, age-related macular degeneration (ARMD), glaucoma and diabetic retinopathy.

Screening for visual acuity

Although people may notice reduced visual acuity while reading, watching television, recognizing people or in other activities, surprisingly such deficits often go unrecognized by the people and their physicians. In one study, up to one third of elderly day patients were found to have unrecognized severe visual loss,[7] and in another, one quarter were wearing inappropriate corrective lenses.[8]

The question "When wearing glasses, can you see well enough to recognize a friend across the street?" had a sensitivity of 48% in detecting visual acuity of less than 20/40 (6/12).[9] Other questions have been found to have a sensitivity of only 20% to 30%.[10] Impaired visual acuity is readily detected with the use of a Snellen sight chart, which can be used in a family physician's office or, in a reduced size, as a portable tool.

A portable visual acuity box was used in the Visual Acuity Impairment Survey Pilot Study.[11] Nearly 1200 subjects were screened, and 123 (10.5%), mostly elderly people, were found to have poor acuity of some degree. Less than half of the subjects were then examined at an eye clinic. Compared with the clinic examination, the portable unit had a sensitivity of 94% and a specificity of 89%. Lowenstein and associates[12] found that when the sight chart was viewed through a pinhole (which minimizes refractive error) the sensitivity was 79% and the specificity 98%.

In a study in Wales 202 elderly patients attending an outpatient clinic were questioned about their use of eyewear and their vision and then were tested with the use of a Snellen sight chart viewed through a pinhole.[13] Over one third of the patients were found to have impaired vision; 30 had refractive errors. Of the 42 patients with nonrefractive errors 27 had a treatable condition (most often cataracts or glaucoma) discovered by an ophthalmologist, and 15 had an untreatable, serious condition (usually ARMD). Only 9 of the 42 believed that their vision was inadequate.

Over 400 consecutive patients attending a primary care general medical clinic in Baltimore were asked to complete a brief questionnaire and undergo a standard vision test with a Snellen sight chart.[14] Nearly two thirds did not meet predefined criteria and were referred to an ophthalmology clinic. Of the 101 who showed up for the evaluation at the clinic, 96 were found to have serious eye disease, the most prevalent being cataracts, diabetic retinopathy, glaucoma and ARMD. Immediate medical therapy was required for 14% and surgical intervention for 18%. The vision test alone failed to identify most cases of diabetic retinopathy and glaucoma.

These case series illustrate the ability of simple questions and visual acuity testing to detect significant ophthalmic disease. Lack of complete follow-up in these studies precludes calculation of test characteristics for these manoeuvres.

Major causes of visual impairment

Presbyopia

As people get older their lenses become thicker and less flexible, which results in diminishing accommodation and often refractive errors. The process is universal with aging but does not usually result in blindness.

Burden of illness

Although the prevalence of visual impairment from all causes is known, that of presbyopia alone is not available. Among 5300 people screened in the Baltimore Eye Survey the prevalence of visual impairment rose from 1.1% among white people aged 60 to 69 years to 14.6% among white people over 80; the corresponding figures among black people were 3.4% and 18.0%.[15] The respective rates for legal blindness (20/200 [6/60]) were 0.3% and 11.6% among the white people and 3.1% and 10.0% among the black people.

Effectiveness of intervention

Refractive errors due to presbyopia are readily corrected with eyeglasses or contact lenses. In the Baltimore Eye Survey visual acuity was measured with the person wearing his or her corrective lenses, if any. Following appropriate correction 54.0% of the subjects had improved vision by at least one line on the Snellen sight chart, and 7.5% had improved vision by three or more lines.

Cataract

The presence of any opacity in the lens is defined as cataract. Cataracts appear in different forms and sizes and may result from trauma, disease (e.g., diabetes or hypoparathyroidism), ionizing radiation or the use of medications (e.g., corticosteroids or antineoplastic agents). In most cases they are idiopathic or "senile."

Burden of illness

The prevalence of cataracts is age dependent. That of cataracts sufficient to impair visual acuity to less than 20/30 (6/9) has been found to increase from 1% among people in their 40s to 100% among those in their 80s.[16–19] Cataracts account for 15% of cases of blindness in Canada[20] and 4.5% to 8.2% of new cases.[6] In the Baltimore Eye Survey blindness was caused by cataracts among 13% of elderly white subjects and among 39% of elderly black subjects.[5]

Detection manoeuvre

Symptoms include deterioration in visual acuity, increased glare in bright light and a "halo" seen around objects. In hyperopic patients "second sight," a temporary improvement, results from a myopic shift with the onset of cataract formation. Cataracts are readily detected by

means of ophthalmoscopy. The test characteristics of examination in primary care are unknown.

Effectiveness of intervention

Medical treatment (pupil dilatation) may improve vision around a small central cataract. Definitive treatment requires surgical removal of the cataract. This procedure effectively restores vision, provided the retina functions well and refraction is adequate. Dense cataracts may obscure the presence of retinal disease (e.g., ARMD), which may affect the surgical outcome. The refractive error induced by aphakia can be corrected with the use of eyeglassses or contact lenses or with intraocular lens (IOL) implantation. IOL implantation is frequently performed during surgery to remove the cataract, and visual correction is far superior to that provided by eyeglasses, which produce substantial distortion. Increasingly, outpatient cataract surgery is being undertaken.

Lens removal with IOL implantation improves vision in approximately 90% of patients;[21] 80% obtain a final visual acuity of 20/40 (6/12) or better. In 5% acuity worsens, and in another 5% it is unchanged.[22] In one study, before IOL implantation 75% of patients using eyeglasses complained about their quality of vision.[23] The degree of patient satisfaction with IOL implantation has been found to be similar to that with extended-wear contact lens.[24] After undergoing cataract surgery with IOL implantation nearly 300 elderly patients reported improvement in their vision, their ability to read newspapers, to drive and to perform daily activities, and their economic resources. The mean improvement in acuity was from 20/100 (6/30) to 20/40 (6/12). Objective improvement was evident in mental status, writing and fine-motor control.[25]

Complications of cataract surgery with IOL implantation include infection, which occurs in up to 3% of cases.[26] Macular edema occurs as a late complication in about 4%, retinal detachment in about 2% and lens dislocation in about 1%. Opacification of the posterior capsule, most commonly seen in posterior-chamber IOL implantation, occurs in up to 5% of cases and may be treated with laser capsulotomy.[27]

Ophthalmologists usually offer cataract surgery when an otherwise healthy patient senses a significant impairment in daily life caused by the vision loss. It is prudent to consult an ophthalmologist early in the cataract progression for a thorough examination of the retina. Retinal examination may be more difficult with mature or "ripened" cataracts.

AGE-RELATED MACULAR DEGENERATION

ARMD is a leading cause of blindness, accounting for 40% to 50% of new cases of blindness in Canada in the last 5 years.[6] It causes moderate to severe loss of central vision. Light-generated metabolic waste products accumulate in the retinal pigment epithelium (RPE), which results in degeneration and atrophy of the choroid capillaries. Drusen (pale yellow spots on the retina due to hyaline in Bruch's membrane) are invariably present and usually precede visual disturbances by many years.

There are two forms of ARMD. Atrophic ("dry") macular degeneration rarely results in a visual acuity worse than 20/80 (6/24). Exudative ("wet") or disciform macular degeneration is less frequent but potentially far more devastating. In this type a fragile subretinal neovascular membrane develops that can distort the macula, which results in metamorphopsia (distortion of vision) and may lead to severe and sudden visual loss from retinal detachment. Approximately 90% of people with ARMD have atrophic maculopathy; of those who are legally blind (visual acuity of less than 20/200 [6/60]) about 90% have exudative maculopathy.[28]

Burden of illness

The prevalence of ARMD is about 1% among people who are 55 years of age and increases to 15% among those who are 80.[16] The prevalence of macular changes (presence of any drusen) increases to 35% by age 64 and to 50% by age 85.[29] Risk factors include hyperopia, a family history of ARMD, smoking, blue eyes and chemical exposure at work.[28,30] ARMD is far more prevalent among white Americans than among black Americans.[5]

Natural history

Several factors identify people with drusen alone who are at greatest risk for exudative maculopathy or potentially serious macular changes. In one retrospective survey of 71 patients with bilateral macular drusen 15% had signs of neovascular change by 5 years and nearly 13% experienced severe visual loss.[31] Concurrent pigmentary changes and confluence of drusen increase the likelihood of neovascular change. Among people with one eye affected by exudative degeneration, the annual incidence of similar change in the other eye has been 12% to 15%.[32,33] Although drusen are the sine qua non of ARMD, not all people with drusen will experience visual loss.

Detection manoeuvre

Distorted near vision (in 29 cases) and blurred vision (in 22 cases) were the most frequent first symptoms among 103 people with ARMD who had recent vision loss from neovascular membranes.[34,35]

Funduscopy reveals drusen and fine pigment stippling in the macular area, which progresses to larger clumps of

pigment. Although drusen are readily recognized by ophthalmologists and optometrists, sensitivity for their detection by primary care physicians is unknown. Most fundi in people over 20 years of age will show drusen histologically; however, only one third of those examined clinically in people over 52 were found to have drusen.[28] The presence of many drusen, pigmentary changes or "softening" should be detectable by primary care physicians.

Retinal photography is a standardized way to record abnormalities of the fundus, but it is not available to primary care physicians. Fluorescein angiography evaluates the vascularity of the fundus and determines capillary leakage, but it is unsuitable for screening.

The Amsler chart is a grid 10 × 10 cm containing 20 5-mm squares to a side, with white lines on a black background. The patient is asked to view it daily to detect metamorphopsia, which indicates early retinal detachment, for which immediate treatment may be beneficial. Although the use of Amsler's chart has been described in case reports[33] compliance is often poor. Of 103 patients with recent onset of visual loss from neovascular maculopathy, 89 were given the chart, but only 49 used it regularly. All but 1 of the 49 had a demonstrable grid defect at the time of diagnosis; however, only 5 noticed the first visual symptom while observing the chart.[34] A modified Amsler's chart the size of a credit card has been found to detect metamorphopsia as well as the original model, but its use in practice has not been evaluated.[36] A suggested (but unproven) alternative is to ask patients at risk to observe carefully a rectangular object (e.g., a window or doorframe) daily to detect metamorphopsia.

Effectiveness of intervention

There was no effective treatment for ARMD before the introduction of laser photocoagulation. Bursts of argon (blue-green) laser energy are used to obliterate the neovascular changes. In three randomized controlled trials photocoagulation was compared with no treatment among subjects over 50 years of age with drusen and subretinal neovascular complexes identified by means of fluorescein angiography.[37–40] In each study, photocoagulation improved preservation of visual acuity. Older patients and those with neovascular tissue distant from the fovea were the most likely to benefit.

The benefits of photocoagulation offer a rationale for early detection and observation of ARMD. Unfortunately, visual deterioration usually occurs and lesions progress beyond the point at which treatment is successful. For example, in one study the condition was treatable in 80% of patients who presented to an ophthalmologist within 2 weeks after symptoms developed; this rate dropped to 40% among those who presented after 1 month and to less than 10% among those who waited 4 months.[41] Disciform ARMD may be amenable to treatment in up to 50% of patients if it is identified early enough. Treatment is most beneficial for patients with a visual acuity of 20/60 (6/18) or better. In the event of retinal detachment, there is no benefit to photocoagulation.[38] Photocoagulation using krypton red laser energy has been found to be no more effective than that using argon green laser energy.[42,43]

Glaucoma

Glaucoma is characterized by increased intraocular pressure (IOP), atrophy of the optic nerve and visual field defects, which usually begin midperipherally. Diagnosis requires two of the three factors to be present. IOP without the other two factors does not indicate glaucoma but does identify people with ocular hypertension and those at risk for glaucoma. Glaucoma may occur with normal IOP (low-tension glaucoma). Open-angle glaucoma is most common (in 90% of cases) and is initially asymptomatic.

Burden of illness

Prevalence studies are complicated by variable diagnostic criteria. Table 1 shows the prevalence of increased IOP in four populations and the proportion discovered to have glaucomatous visual field defects. Table 2 shows the prevalence of glaucoma in six populations. In the Beaver Dam Eye Study,[50] involving over 4000 people residing in a single community, the age-specific prevalence

Table 1: Prevalence of increased intraocular pressure (IOP) and glaucomatous visual field defects

Study and location	Age range of subjects, yr	IOP, mm Hg	Prevalence of increased IOP, %	% of subjects with visual field defects	Ratio of field defect to increased IOP
Hollows et al,[44] Ferndale, Wales	40–74	> 20	9.4	0.31	1:30
Bengtsson,[45] Dalby, Sweden	55–70	> 20.5	7.3	0.33	1:22
Liebowitz et al,[46] Framingham, Mass.	52–85	> 21	7.6	0.36	1:21
Stromberg,[47] Skovde, Sweden	> 40	> 21	4.5	0.36	1:12

of glaucoma rose from less than 0.5% among people 50 years old to 2.5% among 75-year-old men and nearly 7% among 75-year-old women.

Natural history

An IOP of 21 mm Hg is the accepted upper limit of normal; however, glaucoma does not develop in many people with higher pressures and may develop in those with an IOP below 21 mm Hg. Among people over 60 years old, up to 3.7% of those with an IOP of 16 to 19 mm Hg will have glaucomatous visual field defects in 5 years;[51] of those with an IOP of 20 to 23 mm Hg, up to 9.3% will have such defects within 5 years. Thus, more cases of glaucoma will develop in people with an IOP of less than 21 mm Hg than in those with a pressure above 21 mm Hg. The sensitivity of the optic nerve to any given level of pressure determines progression to glaucoma. Risk factors for progression include age, IOP, diabetes, myopia, black race and vascular problems (e.g., hypertension). In people with frank glaucoma, there is some evidence that visual field loss is directly related to IOP and that eyes showing the fastest rate of loss are in an earlier stage of the disease.[52]

Detection manoeuvre

The earliest symptom of open-angle glaucoma is loss of peripheral vision, which frequently goes unnoticed. Acute deterioration of vision, with pain and redness of the orbit, is characteristic of acute angle-closure glaucoma, a condition that is much less common than open-angle glaucoma.

There are three methods of detecting chronic open-angle glaucoma: tonometry, inspection of the optic disc and perimetry.

Tonometry

Tonometry, or measurement of the IOP, is often carried out with the use of a Schiötz tonometer, which estimates pressure by indicating the ease with which the cornea is indented. Tonometry is relatively cheap and accessible to primary care physicians. Applanation tonometry requires a slit lamp, although a hand-held model (Perkins) is now available. Puff tonometry uses a jet of air to deform the cornea.

Readings do not accurately reflect the presence or absence of glaucoma, since normal pressure shows diurnal variations of as much as 5 mm Hg. Among people with glaucoma, variations may be as high as 8 to 10 mm Hg, and only 50% have elevated IOP in random measurement.[53] Biologic factors (position of the patient, presence of myopia, systemic drug use, variation between seasons) and position of the tonometer increase variability. In one study the sensitivity of the Schiötz tonometer was only 50%;[44] the positive predictive value for diagnosing glaucoma has been found to be only 2% to 5%.[44,53,54] Although Schiötz tonometry has been recommended in the past, poor sensitivity, low prevalence of glaucoma and the increasing ratio of elevated IOP to glaucoma with age are grounds to recommend against its sole use in screening.

Inspection of the optic disc

An experienced ophthalmoscopist is able to recognize an increased optic cup–disc ratio in excess of 60%.[55] For ophthalmologists, the sensitivity and specificity of this sign can each exceed 90% for the diagnosis of glaucoma, although clinical disagreement occurs even with predefined criteria.[56,57] It is unlikely that this diagnostic accuracy could be equalled by family physicians. In a study involving 22 patients, 34 ophthalmologists, using funduscopy alone, detected glaucoma in only about half of the cases;[58] there was no evidence that greater experience would improve this rate.

Perimetry

Some 30% to 50% of the optic nerve fibres must be lost before a classic glaucomatous visual field defect con-

Table 2: Prevalence of glaucoma

Study and location	Age range of subjects, yr	Diagnostic methods*	Prevalence of glaucoma, %	% of subjects with normal IOP
Hollows et al,[44] Ferndale, Wales	55–70	T, D, VFD	0.47	35
Liebowitz et al,[46] Framingham, Mass.	52–85	T, VFD	1.43	53
Stromberg,[47] Skovde, Sweden	> 40	T, D, VFD	0.41	13
Stromberg,[47] Dalby, Sweden	> 40	T, D, VFD	0.86	62
Bankes et al,[48] Bedford, England	> 40	T, D, VFD	0.76	7
Armaly,[49] Des Moines, Iowa	20–89	T, D, VFD	4.08	68

*T = tonometry, D = inspection of optic disc, VFD = detection of visual field defect by perimetry.

sistently occurs. Perimetry has been used as a screening test for glaucoma. However, the equipment is costly and not generally available to primary care physicians. It has a high sensitivity for detecting loss of peripheral vision but has a low specificity for glaucoma. Automated visual field screening is feasible and may be practical in the future.[59] Humphrey automated perimetry, which takes about 30 minutes to perform, was found to have a sensitivity of 90% and a specificity of 91% when compared with the standard Goldmann perimetry device.[60]

Effectiveness of intervention

Visual loss in glaucoma is not generally reversible, although some improvement in the field of vision may occur in the first 6 months of topical treatment.[61] Treatment is aimed at reducing IOP with the use of topical agents (e.g., pilocarpine or ß-adrenergic blockers [e.g., timolol and betaxolol]), systemic agents (e.g., acetazolamide) or surgery.

Although it is well accepted that reducing extremely high IOP (e.g., above 35 mm Hg) prevents visual loss, such levels rarely occur in the general population. The benefit of treating mild to moderate IOP is less clear. In seven randomized controlled trials of the benefit of lowering the IOP, the development of new visual field defects was measured by means of perimetry.[62–69] The recent studies used automated perimetry, which minimized observer bias. Although five of the studies showed a benefit,[62,64–68] two,[63,69] including the largest and most recent,[69] did not. The studies' methodologic differences make it difficult to draw definite conclusions. A recent meta-analysis of these studies concluded that the odds of progression to visual field deficits was reduced by 25%, but a wide 95% confidence interval (0.43 to 1.34) implied that the risk of worsening deficits could not be excluded.[70]

Systemic agents such as acetazolamide also reliably reduce IOP.[71] Side effects include hypokalemia and metabolic acidosis. Surgical treatment is effective in lowering IOP but is usually indicated only when medical treatment fails.[72] Argon laser trabeculoplasty has been introduced as an alternative treatment to topical medication in people with primary open-angle glaucoma. In a study involving 271 patients one eye was treated with laser therapy and the other with timolol eye drops.[73] After 2 years, the visual acuity and fields did not differ significantly, but fewer laser-treated eyes required two or more medications to control IOP.

Diabetic retinopathy

Diabetic retinopathy is a chronic disorder of the microcirculatory system of the retina. Background retinopathy occurs in both type I (ketosis-prone, insulin-dependent) and type II (non-ketosis-prone, usually non-insulin-dependent) diabetes mellitus and is recognized clinically as microaneurysms and "dot" or "blot" hemorrhages. Maculopathy is the commonest cause of visual impairment in patients with diabetic retinopathy and is more common in patients with type II diabetes. Proliferative retinopathy is due to the formation of new vessels in ischemic areas of the retina. It is more common in patients with type I diabetes than in those with type II disease and may result in blindness because of hemorrhage or retinal disruption.

Burden of illness

In older diabetec patients retinopathy accounts for 33% of cases of blindness.[74] In the Framingham Eye Study the 4-year incidence of blindness (visual acuity of less than 20/200 [6/60]) was about 3% among older subjects taking insulin or not.[75] By 20 years virtually all of the patients with type I diabetes and 60% of of those with type II diabetes are expected to have some degree of retinopathy. The 4-year incidence of macular edema among older people was about 8% among those using insulin and 3% among those not using insulin.[76] Risk factors for retinopathy include hypertension, poor control of diabetes, duration of diabetes, heavy alcohol consumption and cigarette smoking.

Detection manoeuvre

The WHO defines impaired carbohydrate tolerance as a blood sugar level of 7.8 mmol/L after fasting or 11.1 mmol/L 2 hours after eating.[3]

Background retinopathy and advanced proliferative retinopathy should be detectable by family physicians. In a study in which funduscopy was compared with retinal photography among patients known to have diabetes, signs of proliferative retinopathy were detected by all of the retinal subspecialists; however, 49% of these changes were missed by internists, diabetologists and senior medical residents.[77] In another study optometrists diagnosed the presence or absence of retinopathy in 77% of patients under ideal conditions.[78] In a study involving more than 2000 people with diabetes, there was exact agreement in categorizing retinopathy as absent, proliferative or nonproliferative between ophthalmologists, specially trained optometrists and ophthalmic technicians in 85.7% of cases (kappa 0.749).[79] The ability to detect serious retinopathy is highly dependent on technique and experience. Without pupillary dilatation the sensitivity of ophthalmoscopy was found to be between 38% and 50% when performed by diabetologists or experienced technicians.[80] With dilatation it was 70%

and 82% when performed by diabetologists and retinal specialists respectively. When carried out by nurses the sensitivity was 0% and the specificity 100%.[80] The gold standard in these studies was a seven-field stereoscopic photograph of the fundus. There has been interest in nonmydriatic fundus photography with a single 45° view of the posterior pole of each eye. This technique compares favourably with ophthalmoscopy with dilatation and is considerably more accurate than ophthalmoscopy without dilatation by a trained observer.[80–83]

Retinal photography requires costly equipment. Because diabetic retinopathy is common, further evaluation is required to determine whether this technique is cost effective in screening people with diabetes.

Effectiveness of intervention

Although tighter metabolic control delays most diabetic complications, one study comparing regular treatment with intensified diabetic control (continuous subcutaneous insulin therapy) revealed that retinopathy appeared to accelerate initially in the latter group.[84] After 2 years the degree of retinopathy was indistinguishable between the two groups, with a trend toward lesser overall deterioration in the continuous insulin group.

Xenon arc and argon laser photocoagulation maintain vision and reduce the risk of visual loss associated with various types of diabetic retinopathy. In cases of proliferative retinopathy two major studies have confirmed the benefit of photocoagulation.[85–87] Xenon arc and argon laser photocoagulation have also been found to reduce the risk of visual loss in people with diabetic macular edema.[88,89] The best results occurred in people whose initial vision was good (20/20 [6/6] to 20/30 [6/9]). Medical treatment with sorbinil or acetylsalicylic acid has proven ineffective.[90–92]

Proliferative retinopathy affects patients with either type of diabetes, and its incidence depends on the duration of the diabetes. Treatment with photocoagulation is effective in preserving sight. Screening patients with type I diabetes beginning 5 years after the onset of the diabetes has been calculated to produce an average expected benefit of 2.7 person-years of sight saved per person screened, with a net saving of medical and social security costs of about $3300 (US) per person screened.[93,94] For patients with type II diabetes not taking insulin, it was estimated that each year of sight saved would cost $1500 (US).[95] Screening of patients with insulin-dependent diabetes, type I or type II, is cost saving. Screening with fundus photography was slightly more effective than that with ophthalmoscopy.[95]

Recommendations (Table 3)

It is uncertain how many elderly Canadians receive regular eye care, or from whom. Family physicians traditionally provide primary eye care. Many elderly people, particularly in urban centres, receive regular eye care from ophthalmologists. Optometrists are distributed throughout urban and rural Canada.

Underreporting of visual impairment is common in the elderly population. Testing for visual acuity has been widely recommended. Although many abnormalities will be discovered, there is scant evidence that correction of poor visual acuity will improve function or quality of life or reduce hazards of daily life.

Visual impairment due to presbyopia and other refractive errors is common in elderly people, and its prevalence increases with age. Visual loss is often underreported. Detection is straightforward, and useful improvements in vision frequently result from refraction and corrective eyewear.

Notwithstanding the lack of evidence of benefit, there is no reason to believe that screening or case finding for visual impairment would be harmful. Given the high prevalence rates and effective treatment, there is fair evidence to include visual acuity testing with a Snellen sight chart in the periodic health examination of elderly patients (grade B recommendation). The optimal frequency of testing is uncertain.

Tests of visual acuity will not detect early signs of exudative ARMD or of macular edema from diabetic retinopathy. Both conditions are detectable by means of funduscopy and are likely treatable with photocoagulation; patients with better visual acuity are most likely to benefit. The accuracy of primary care physicians in diagnosing these conditions is unknown. A British survey showed that referrals to ophthalmologists from ophthalmic opticians (optometrists) were more appropriate and showed more highly developed funduscopic skills than referrals from family physicians.[98] Whether the initial assessment should be done by a primary care physician or by an optometrist is uncertain, as is the optimal frequency of re-examination. Until the characteristics of retinal examination by primary care physicians have been better defined, there is insufficient evidence to include funduscopy in or exclude it from the periodic health examination (grade C recommendation). Since diabetic retinopathy and ARMD may have dire consequences and there is good evidence of effective treatment, the prudent physician may wish to include funduscopy in the periodic health examination. If ARMD is detected, patients should be referred to an ophthalmologist.

Cataracts are common and cause progressive visual loss, which can be effectively treated by means of cataract removal and corrective lenses. Early detection and referral to an ophthalmologist may facilitate assessment of retinal disease before the fundus is obliterated by advancing cataract; however, most patients will complain of visual loss, and primary care physicians can

readily identify cataract and arrange for referral when visual problems occur. Thus, there is no convincing rationale for early detection.

Annual screening for diabetic retinopathy is recommended for all patients with type I diabetes who have had the disease for at least 5 years. For patients with type II diabetes the same recommendation can be made; however, if seven-field stereoscopic photographs of the fundus show no signs of retinopathy at the time of diagnosis, screening may be delayed for 4 years.[96] Examination is preferably carried out by an ophthalmologist, but if this is not possible, funduscopy with dilatation or fundus photography are alternatives.

Although open-angle glaucoma is a relatively common cause of visual loss among older people, there are problems with the screening manoeuvres. Automated perimetry, a new technique, shows promise but has not been widely evaluated in community surveys. Treatment of isolated IOP probably prevents visual field loss and provides the rationale for screening with tonometry. Because of the poor sensitivity and low positive predictive value of Schiötz tonometry, this technique is not recommended for screening.[99] Other methods of measuring IOP (e.g., Perkins tonometry or puff tonometry) are now available, and a high research priority is to determine the performance of these methods in community screening. The calculated average cost per year of vision saved is lower for ophthalmoscopy than for tonometry with increasing age.[100] Screening with automated perimetry is the most cost-effective approach among people over 65 years old.[70] Detection of visual field loss may be the preferred method of screening for glaucoma in future.[59] Older people who have a family history of glaucoma, are black, have severe myopia or have diabetes are at greatest risk of glaucoma. A prudent recommendation is to include periodic assessment by an ophthalmologist who has access to automated perimetry; the optimal interval is uncertain.

VALIDATION

The background document prepared by the principal author (C.P.) was reviewed by the members of the task force on several occasions and by several external re-

Table 3: Summary of manoeuvres, effectiveness, levels of evidence and recommendations for screening for visual impairment among elderly patients

Manoeuvre	Effectiveness	Level of evidence*	Recommendation*
Snellen sight chart	Use of chart reliably detects reduced visual acuity in community studies	Cohort study[11,12] (II-2)	Fair evidence to include in the periodic health examination (PHE) (B)
	Population screening can lead to useful improvements in vision	Cohort study[15] (II-2)	
Funduscopy or retinal photography in diabetic patients	Funduscopy and retinal photography are sensitive for detecting diabetic retinopathy; early detection preserves vision	Expert opinion[96] (III)	Fair evidence to include in the PHE of diabetic patients (B)
	Photocoagulation in proliferative diabetic retinopathy preserves vision	Randomized controlled trials[85-87,89] (I)	
Funduscopy to detect age-related macular degeneration (ARMD)	ARMD can be detected by those trained in ophthalmoscopy	Expert opinion[38] (III)	Insufficient evidence to include in or exclude from the PHE (C)
	Photocoagulation preserves vision in patients with neovascular changes from ARMD	Randomized controlled trials[37-40] (I)	
Funduscopy, tonometry or automated perimetry to detect glaucoma	Examination of optic disc (funduscopy) is sensitive for detecting glaucoma	Cohort study[97] (II-2)	Insufficient evidence to include in or exclude from the PHE (C)
	Schiötz tonometry has poor sensitivity and specificity for early detection of glaucoma	Case series[42,53,54] (III)	
	Automated perimetry (Humphrey) is sensitive for detecting glaucoma	Case series[60] (III)	
	Topical application of ß-adrenergic blocker lowers IOP and may retard vision loss	Randomized controlled trials[62-70] (I)	

*For descriptions of the other levels of evidence and classification of recommendations see Appendix 1 in part 1 of the 1992 update (*Can Med Assoc J* 1992; 147: 443).

viewers. The recommendations differ from those of the American Academy of Ophthalmology and the American Optometric Association. Those for glaucoma screening are similar to the recommendations of the US Preventive Services Task Force. The following highlights recommendations from other sources.

- The American Academy of Ophthalmology recommends that ophthalmoscopy and tonometry be performed annually among all people over age 40.[101] A complete ocular examination by an ophthalmologist is recommended at least once between the ages of 35 and 45 and should be repeated every 5 years after age 50.
- The American Optometric Association recommends a complete eye and vision examination, including tonometry, for people over 35 (D. James, American Optometric Association: personal communication, 1993).
- The US Preventive Services Task Force suggests advising people at high risk for glaucoma (e.g., those aged 65 or more) to be tested by an eye specialist. The optimal frequency is left to clinical discretion.[99]
- Schiötz tonometry should no longer be recommended as a technique for the early detection of glaucoma.[102]

Research priorities

1. Determination of which simple questions have the best sensitivity for detecting visual problems.
2. Determination of the sensitivity and specificity of the Snellen sight chart for detecting visual impairment by primary care physicians.
3. Evaluation of the value of funduscopy in predicting pressure-induced ocular damage.
4. Determination of the characteristics of funduscopy in the primary care setting for detecting ARMD.
5. Exploration of the most effective method of improving the funduscopic skills of primary care physicians.
6. Comparison of the cost-effectiveness of providing currently available automated visual-field screening devices to primary care physicians and of training them to reliably recognize the funduscopic characteristics of a glaucomatous optic disc.
7. Determination of the most effective method of detecting glaucoma. Evaluating the performance of puff tonometry and Perkins tonometry in community screening.
8. Exploration of the role of optometrists in primary care screening for visual problems.

We thank ophthalmologists Vladamir Kozousek, Camp Hill Hospital, Halifax, Michael A. Motolko, Toronto Hospital, and Graham E. Trope, chair, Department of Ophthalmology, University of Toronto, and optometrists Rodger Pace, School of Optometry, University of Waterloo, Waterloo, Ont., and Raymond Wagg, president, Nova Scotia Association of Optometrists, New Glasgow, NS, for their careful reviews and advice in the preparation of this manuscript.

The task force is funded by the Health Services and Promotion Branch, Health Canada, and by the National Health Research Development Program (grants 6605-2702-57X and 6603-1375-57X). Dr. Christopher Patterson was supported in part by the Educational Centre for Aging and Health, McMaster University, Hamilton, Ont.

References

1. Canadian Task Force on the Periodic Health Examination: The periodic health examination. *Can Med Assoc J* 1979; 121: 1193–1254
2. Riordan-Eva P: Blindness. In Vaughan D, Asbury T, Tabbara KF (eds): *General Ophthalmology*, 12th ed, Appleton and Lange, Norwalk, Conn, 1989: 384–388
3. *The Prevention of Blindness* (no 518, Technical Report series), World Health Organization, Geneva, 1973
4. Nelson KA: Visual impairment among elderly Americans: statistics in transition. *J Vis Impair Blind* 1987; 81: 331–334
5. Sommer A, Tielsch JM, Katz J et al: Racial differences in the cause-specific prevalence of blindess in East Baltimore. *N Engl J Med* 1991; 325: 1412–1417
6. *Statistical Information on the Client Population of the CNIB*, Canadian National Institute for the Blind, Toronto, 1989
7. McMurdo MET, Baines PS: The detection of visual disability in the elderly. *Health Bull* 1988; 46: 327–329
8. Stults BM: Preventive health care for the elderly. *West J Med* 1984; 141: 832–845
9. Stone DH, Shannon DJ: Screening for impaired visual acuity in middle age in general practice. *BMJ* 1978; 2: 859–863
10. Hiller R, Krueger DE: Validity of a survey question as a measure of visual acuity impairment. *Am J Public Health* 1983; 73: 93–96
11. Ederer F, Krueger DE, Mowery RL et al: Lessons from the Visual Acuity Impairment Survey Pilot Study. *Am J Public Health* 1986; 76: 160–165
12. Lowenstein JI, Palmberg PF, Connett JE et al: Effectiveness of a pinhole method for visual screening. *Arch Ophthalmol* 1985; 103: 222–223
13. Long CA, Holden R, Mulkerrin E et al: Opportunistic screening of visual acuity of elderly patients attending outpatient clinics. *Age Ageing* 1991; 20: 392–395
14. Strahlman E, Ford D, Whelton P et al: Vision screening in a primary care setting. A missed opportunity? *Arch Intern Med* 1990; 150: 2159–2164
15. Tielsch JM, Sommer A, Witt K et al: Blindness and visual impairment in an American urban population. The Baltimore Eye Survey. *Arch Ophthalmol* 1990; 108: 286–290
16. Podger MJ, Leske MC, Ederer F: Incidence estimates for lens changes, macular changes, open angle glaucoma and diabetic retinopathy. *Am J Epidemiol* 1983; 118: 206–212
17. McGuinness R: The Framingham Eye Study. *Am J Ophthalmol* 1978; 86: 852–853
18. Kahn HA, Moorhead HB: *Statistics on Blindness in the Model Reporting Area, 1969–1970* (PHS publ no [NIH] 73-427), US Department of Health, Education, and Welfare, Washington, 1973
19. Johasson F, Thordarson K Prevalence of ocular disease and blindness in a rural area in the Eastern Region of Iceland dur-

ing 1980 through 1984. *Acta Ophthalmol* 1987; 65 (suppl 182): 40–43
20. Leske MC, Sperduto RD: The epidemiology of senile cataracts: a review. [review] *Am J Epidemiol* 1983; 118: 152–165
21. Straatsma BR, Meyer KT, Bastek JV et al: Posterior chamber intraocular lens implantation by ophthalmology residents. *Opthalmology* 1983; 90: 327–335
22. Miller ST, Graney MJ, Elam JT et al: Predictions of outcomes from cataract surgery in elderly persons. *Ophthalmology* 1988; 95: 1125–1129
23. Bernth-Petersen B: Outcome of cataract surgery I. *Acta Ophthalmol* 1982; 60: 235–242
24. Immonen I, Tuominen R, Raivio I: Visual results and social rehabilitation after cataract surgery. *Acta Ophthalmol* 1988; 66: 572–576
25. Applegate WB, Miller ST, Elam JT et al: Impact of cataract surgery with lens implantation on vision and physical function in elderly patients. *JAMA* 1987; 257: 1064–1066
26. Carlson AN, Tetz MR, Apple D: Infectious complications of modern cataract surgery and intraocular lens implantation. *Infect Dis Clin North Am* 1989; 3: 339–355
27. Lorusso V, Moramarco A, Pacella E et al: Intraocular lens complications. *Ann Ophthalmol* 1990; 22: 377–381
28. Ferris FL: Senile macular degeneration: review of epidemiological features. *Am J Epidemiol* 1983; 118: 132–151
29. Sperduto RD, Seigel D: Senile lens and senile macular changes in a population-based sample. *Am J Ophthalmol* 1980; 90: 86–91
30. Hyman LG, Lilienfeld AM, Ferris FL et al: Senile macular degeneration: a case–control study. *Am J Epidemiol* 1983; 118: 213–227
31. Smiddy WE, Fine SL: Prognosis of patients with bilateral macular drusen. *Ophthalmology* 1984; 91: 271–277
32. Gregor Z, Bird AC, Chisholm IH: Senile disciform macular degeneration in the second eye. *Br J Ophthalmol* 1977; 61: 141–147
33. Bressler SB, Bressler NM, Fine SL et al: Natural course of choroidal neovascular membranes within the foveal avascular zone in senile macular degeneration. *Am J Ophthalmol* 1982; 93: 157–163
34. Fine AM, Elman MJ, Ebert JE et al: Earliest symptoms caused by neovascular membranes in the macula. *Arch Ophthalmol* 1986; 104: 513–514
35. Fine SL: Early detection of extra foveal neurovascular membranes by daily central field evaluation. *Ophthalmology* 1985; 92: 603–609
36. Yannuzzi LA: A modified Amsler grid. *Ophthalmology* 1982; 89: 157–159
37. Macular Photocoagulation Study Group: Argon laser photocoagulation for senile macular degeneration. *Arch Ophthalmol* 1982; 100: 912–918
38. Moorfields Macular Study Group: Retinal pigment epithelial detachments in the elderly: a controlled trial of argon laser photocoagulation. *Br J Ophthalmol* 1982; 66: 1–16
39. Moorfields Macular Study Group: Treatment of senile disciform macular degeneration: a single blind randomised trial by argon laser photocoagulation. *Br J Ophthalmol* 1982; 66: 745–753
40. Coscas G, Soubrane G: Photocoagulation de néovaisseaux sou-rétiniens par le laser à argon dans la dégénérescence maculaire sénile. *Bull Mem Soc Fr Ophtalmol* 1982; 83: 102–105
41. Grey RHB, Bird AC, Chisholm IH: Senile disciform macular degeneration: features indicating suitability for photocoagulation. *Br J Ophthalmol* 1979; 63: 85–89
42. Macular Photocoagulation Group: Laser photocoagulation of subfoveal neovascular lesions in age-related macular degeneration: results of a randomized clinical trial. *Arch Ophthalmol* 1991; 109: 1220–1231
43. Canadian Ophthalmology Study Group: Argon green vs. krypton red laser photocoagulation of extrafoveal choroidal neovascular lesions. *Arch Ophthalmol* 1993; 111: 181–185
44. Hollows FC, Graham PA: Intraocular pressure, glaucoma and glaucoma suspects in a defined population. *Br J Ophthalmol* 1966; 50: 570–586
45. Bengtsson B: The prevalence of glaucoma. *Br J Ophthalmol* 1981; 65: 46–49
46. Leibowitz HM, Krueger DE, Maunder LR et al: The Framingham Eye Study Monograph. *Surv Ophthalmol* 1980; 24 (suppl): 366–610
47. Stromberg U: Ocular hypertension. *Acta Ophthalmol* 1962; 69 (suppl): 7–75
48. Bankes JLK, Perkins ES, Tsolakis S et al: Bedford glaucoma survey. *BMJ* 1968; 1: 791–796
49. Armaly MF: On the distribution of applanation pressure and arcuate scotoma. In Paterson G, Miller SJH, Paterson GD (eds): *Drug Mechanisms in Glaucoma*, Little, Brown and Company, Boston, 1966: 167–189
50. Klein BEK, Klein R, Sponsel WE et al: Prevalence of glaucoma. The Beaver Dam Eye Study. *Ophthalmology* 1992; 99: 1499–1504
51. Armaly MF, Krueger DE, Maunder L et al: Biostatistical analysis of the Collaborative Glaucoma Study. I. Summary report of the risk factors for glaucomatous visual-field defects. *Arch Ophthalmol* 1980; 98: 2163–2171
52. O'Brien C, Schwartz B, Takamoto T: Intraocular pressure and the rate of visual field loss in chronic open-angle glaucoma. *Am J Ophthalmol* 1991; 111: 491–500
53. Leske MC: The epidemiology of open-angle glaucoma: a review. *Am J Epidemiol* 1983; 118: 166–191
54. Ford VJ, Zimmerman TJ, Kooner A: A comparison of screening methods for the detection of glaucoma. [abstract] *Invest Ophthalmol Vis Sci* 1982; 22 (suppl): 257
55. Epstein DL (ed): Examination of the eye in glaucoma. In *Chandler & Grant's Glaucoma*, 3rd ed, Lea & Febiger, Philadelphia, 1986: 74–90
56. Schwartz JT: Methodologic differences and measurement of cup–disc ratio: an epidemiologic assessment. *Arch Ophthalmol* 1976; 94: 1101–1105
57. Holmin C: Optic disc evaluation versus the visual field in chronic glaucoma. Acta Ophthalmol 1982: 275–283
58. Wood GM, Bosanquet R: Limitations of direct ophthalmoscopy in screening for glaucoma. *BMJ* 1987; 294: 1587–1588
59. Bengtsson B: Repeated visual field screening in the aged. *Acta Ophthalmol* 1988; 66: 659–661
60. Trope GE, Britton R: A comparison of Goldmann and Humphrey automated perimetry in patients with glaucoma. *Br J Ophthalmol* 1987; 71: 489–493
61. Messmer C, Flammer J, Stumpfig D: Influence of betaxolol and timolol on the visual fields of patients with glaucoma. *Am J Ophthalmol* 1991; 112: 678–681
62. Becker B, Morton WR: Topical epinephrine in glaucoma sus-

pects. *Am J Ophthalmol* 1966; 62: 272–277
63. Levene RZ: Uniocular miotic therapy. *Trans Am Acad Ophthalmol Otolaryngol* 1975; 79: 376–380
64. Shin DH, Kolker AR, Kass MA et al: Long-term epinephrine therapy of ocular hypertension. *Arch Ophthalmol* 1976; 94: 2059–2060
65. Kitazawa Y: Prophylactic therapy of ocular hypertension: a prospective study. *Trans Ophthalmol Soc NZ* 1981; 33: 30–32
66. Epstein DL, Krug JH, Hertzmark E: A long-term clinical trial of timolol therapy versus no treatment in the management of glaucoma suspects. *Ophthalmology* 1989; 96: 1460–1467
67. Kass MA, Gordon MO, Hoff MR et al: Topical timolol administration reduces the incidence of glaucomatous damage in ocular hypertensive individuals. *Arch Ophthalmol* 1989; 107: 1590–1598
68. Kass MA: Timolol treatment prevents or delays glaucomatous visual field loss in individuals with ocular hypertension: a five-year randomised, double-masked clinical trial. *Trans Am Ophthalmol Soc* 1989; 87: 598–618
69. Schulzer M, Drance SM, Douglas GR: A comparison of treated and untreated glaucoma suspects. *Ophthalmology* 1991; 98: 301–307
70. Rossetti L, Marchetti I, Orzalesi N et al: Randomized clinical trials on medical treatment of glaucoma: Are they appropriate to guide clinical practice? *Arch Ophthalmol* 1993; 111: 96–103
71. Mosteller MW, Zimmerman TJ: The medical management of glaucoma. In Spoor TC (ed): *Modern Management of Ocular Diseases*, Slack Inc, Thorofare, NJ, 1985: 179–188
72. Miller SJH (ed): Glaucoma. In *Parson's Diseases of the Eye*, 18th ed, Churchill Livingstone, Edinburgh, 1990: 226
73. Glaucoma Laser Trial Research Group: The Glaucoma Laser Trial. *Ophthalmology* 1990; 97: 1403–1413
74. Klein R, Klein BEK, Moss SE: Visual impairment in diabetes. *Ophthalmology* 1984; 91: 1–9
75. Moss SE, Klein R, Klein BEK: The incidence of vision loss in a diabetic population. *Ophthalmology* 1988; 95: 1340–1348
76. Klein R, Moss SE, Klein BE et al: The Wisconsin epidemiologic study of diabetic retinopathy. XI. The incidence of macular edema. *Ophthalmology* 1989; 96: 1501–1510
77. Sussman EJ, Tsiaras WG, Soper KA: Diagnosis of diabetic eye disease. *JAMA* 1982; 247: 3231–3234
78. Kleinstein RN, Roseman JM, Herman WH et al: Detection of diabetic retinopathy by optometrists. *J Am Optom Assoc* 1987; 58: 879–882
79. Moss SE, Klein R, Kessler SD et al: Comparison between ophthalmoscopy and fundus photography in determining severity of diabetic retinopathy. *Ophthalmology* 1985; 92: 62–67
80. Singer DE, Nathan DM, Fogel HA et al: Screening for diabetic retinopathy. *Ann Intern Med* 1992; 116: 660–671
81. Forrest RD, Jackson CA, Yudkin JS: Screening for diabetic retinopathy — comparison of a nurse and a doctor with retinal photography. *Diabetes Res* 1987; 5: 39–42
82. Williams R, Nussey S, Humphry R et al: Assessment of non-mydriatic fundus photography in detection of diabetic retinopathy. *BMJ* 1986; 293: 1140–1142
83. Barrie T, MacCuish AC: Assessment of non-mydriatic fundus photography in detection of diabetic retinopathy. [letter] *BMJ* 1986; 293: 1304–1305
84. KROC Collaborative Study Group: Diabetic retinopathy after two years of intensified insulin treatment. Follow-up of the KROC Collaborative Study. *JAMA* 1988; 260: 37–41
85. Diabetic Retinopathy Study Research Group: Preliminary report on effects of photocoagulation therapy. *Am J Ophthalmol* 1976; 81: 383–396
86. Diabetic Retinopathy Study Research Group: Photocoagulation treatment of proliferative diabetic retinopathy. *Ophthalmology* 1981; 88: 583–600
87. British Multicentre Study Group: Proliferative diabetic retinopathy: treatment with xenon arc photocoagulation. Interim report of multicentre randomized controlled clinical trial. *BMJ* 1977; 1: 739–742
88. Early Treatment Diabetic Retinopathy Study Research Group: Photocoagulation for diabetic macular edema. ETDRS report no. 1. *Arch Ophthalmol* 1985; 103: 1796–1806
89. British Multicentre Study Group: Photocoagulation for diabetic maculopathy. A randomized controlled trial using the xenon arc. *Diabetes* 1983; 32: 1010–1016
90. DAMAD Study Group: Effect of aspirin alone and aspirin plus dipyridamole in early diabetic retinopathy. *Diabetes* 1989; 38: 491–498
91. Early treatment diabetic retinopathy study research group: effects of aspirin treatment on diabetic retinopathy. *Ophthalmology* 1991; 98: 757–765
92. Sorbinil Retinopathy Trial Research Group: A randomized trial of sorbinil, an aldose reductase inhibitor, in diabetic retinopathy. *Arch Opthalmol* 1990; 108: 1234–1244
93. Javitt JC, Canner JK, Sommer A: Cost effectiveness of current approaches to the control of retinopathy in type I diabetics. *Ophthalmology* 1989; 96: 255–264
94. Javitt JC, Canner JK, Frank RG et al: Detecting and treating retinopathy in patients with type I diabetes mellitus. A health policy model. *Ophthalmology* 1990; 97: 483–495
95. Dasbach EJ, Fryback DG, Newcomb PA et al: Cost effectiveness strategies for detecting diabetic retinopathy. *Med Care* 1991; 29: 20–39
96. American College of Physicians, American Diabetes Association, and American Academy of Ophthalmology: Screening guidelines for diabetic retinopathy. *Ann Intern Med* 1992; 116: 683–685
97. Cooper RL, Grose GC, Constable IJ: Mass screening of the optic disc for glaucoma: a follow up study. *Aust N Z J Ophthalmol* 1986; 14: 35–39
98. Harrison RJ, Wild JM, Hobley AJ: Referral patterns to an ophthalmic out patient clinic by general practitioners and ophthalmic opticians and the role of these professionals in screening for ocular disease. *BMJ* 1988; 297: 1162–1167
99. US Preventive Services Task Force: *Guide to Clinical Preventive Services: an Assessment of the Effectiveness of 169 Interventions*, Williams & Wilkins, Baltimore, 1989: 187–190
100. Gottlieb LK, Schwartz B, Pauker SG: Glaucoma screening. A cost effectiveness analysis. *Surv Ophthalmol* 1983; 28: 206–226
101. Sommer A, Beauchamp GR, Garcia GE et al: *Comprehensive Adult Eye Evaluation. Preferred Practice Pattern*, American Academy of Ophthalmology, San Francisco, 1989
102. Battista RN, Huston P, Davis MW: Screening for primary open-angle glaucoma. In Goldbloom RB, Lawrence RS (eds): *Preventing Disease: Beyond the Rhetoric*, Springer-Verlag, New York, 1990

For prescribing information see page 1342 →